CHAPTER 10

PERIODONTOLOGY

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STRUCTURE OF THE PERIODONTAL TISSUES

The *periodontium* (*perio* = around, *odontos* = tooth) consist of the tissues which surround and support the teeth. Their function is to attach the tooth to the surrounding alveolar bone and to support the tooth during function. For descriptive purposes the periodontium can be divided into the *gingivae* and *periodontal ligament* (Figure 10.1).

GINGIVAE

Gingivae (singular = gingiva) are those parts of the masticatory mucosa that cover the alveolar process and surround the cervical portion of the teeth.

The gingivae are composed of connective tissue and epithelium, which can be divided into three histologically distinct areas:

Oral epithelium Continuous with epithelial lining of the attached gingivae. Composed of keratinized stratified squamous epithelium.

Sulcular epithelium Non-keratinized.

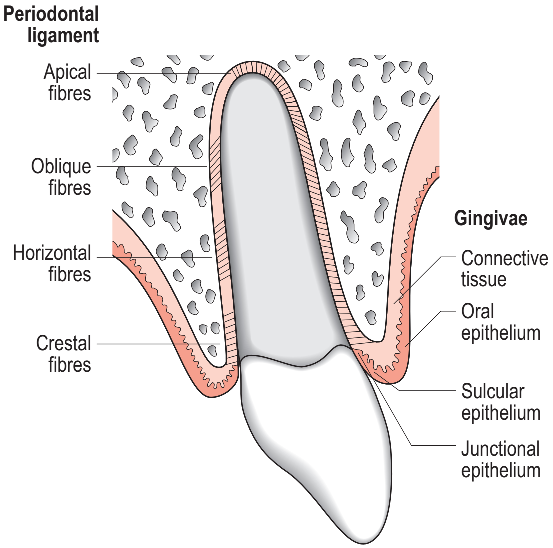


Figure 10.1 Structure of the periodontium.

Junctional epithelium Attached to the tooth by hemidesmosomes. Non-keratinized. Has larger cells with increased intercellular spaces.

Healthy gingivae have the following clinical features: • pale pink in colour • a stippled appearance • a well-defined ‘knife edge’ margin • scalloped outline • do not bleed on gentle probing with a periodontal probe.

PERIODONTAL LIGAMENT

Periodontal ligament is composed of collagen fibres, which form a branching plexus and are inserted into the cementum of the tooth root at one end and the alveolar bone at the other in the form of Sharpey’s fibres. They are named according to the orientation of the fibre.

AETIOLOGY AND PATHOGENESIS OF PERIODONTAL DISEASES

In its widest sense, periodontal disease includes all pathological conditions of the periodontium but commonly refers to inflammatory diseases that are plaque-induced, i.e. *gingivitis* and *periodontitis*.

GINGIVITIS

Gingivitis is an inflammatory response of the gingivae without destruction of the supporting tissues.

In the absence of adequate oral hygiene, dental plaque accumulates and the gingival tissue will progress from health to an established or chronic gingivitis over a 3-week period. The development of gingivitis is encouraged by both local and systemic factors. *Local factors* contributing to the development of gingivitis are those which encourage the accumulation of dental plaque or inhibit effective plaque removal and include calculus, crowded dentition, soft tissue factors, e.g. high fraenal attachment, restorations and prostheses. Systemic factors that influence the host response to plaque accumulation include pregnancy, puberty, diabetes mellitus and blood dyscrasias.

Inflamed gingivae have the following clinical features: • red in colour • swollen appearance • stippling is lost • increased flow of gingival crevicular fluid (GCF) • bleed readily on probing.

Gingivitis can have three possible outcomes, as described in Table 10.1.

PERIODONTITIS

Periodontitis describes a group of inflammatory diseases that affect all the periodontal structures. It results in destruction of the attachment apparatus and development of a periodontal pocket.

The relationship between gingivitis and periodontitis is complex. Whilst plaque accumulation almost always leads to gingivitis, it does not invariably lead to periodontitis. Why only a proportion of sites with gingivitis progress to periodontitis and why this is more likely in some individuals than in others has been the subject of much research.

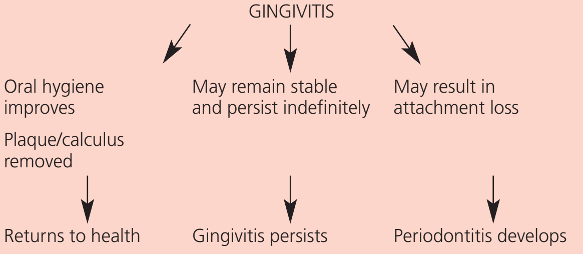
Breakdown of the periodontal ligament and the development of a periodontal pocket is an unpredictable event. When it occurs, the rapidity with which connective tissue attachment is destroyed varies both between individuals and at individual tooth sites within the same mouth.

FACTORS PREDISPOSING TO PERIODONTITIS

Microbiological factors

The precise manner by which plaque organisms induce breakdown of the periodontal tissues is not known. The ‘non-specific plaque

TABLE 10.1 The potential outcomes of gingivitis



hypothesis' postulates that periodontal disease is the result of toxin production by a range of bacterial species and more than 300 species of micro-organisms have been isolated from periodontal pockets. There is evidence, however, that certain Gram-negative anaerobic bacteria are isolated more commonly at sites of active periodontal destruction (Table 10.2). These organisms accumulate as a biofilm and interaction and synergism between organisms is important in the pathogenesis of disease. Virulent micro-organisms capable of initiating or propagating attachment loss, if present at a critical minimal infective dose, in susceptible individuals, or at susceptible periodontal sites in susceptible individuals, have the ability to breach the host defence mechanism, exposing the host tissues to toxic bacterial components. As a result host cells (e.g. monocytes and fibroblasts) are stimulated by bacterial components such as *lipopolysaccharides* (LPS) to produce cytokines. These are powerful chemical messengers which stimulate inflammatory responses and catabolic processes such as bone resorption and collagen destruction via enzymes know as matrix metalloproteinases (MMPs).

Host factors

Bacterial plaque produces many substances which can cause damage to the periodontium. The tissues rely on several host defence mechanisms to protect against plaque irritants. These include: • polymorphonuclear neutrophils (PMNs) • the complement system • the cellular and humoral immune responses • various chemical mediators of inflammation.

In response to plaque accumulation, gingival crevicular fluid (GCF) permeates through the junctional epithelium. This transudate is rich in neutrophils that can neutralize potential pathogenic agents by phagocytosis. It has been suggested that defects in neutrophil function may account for certain forms of periodontal disease. Within the neutrophils, lysosomes (cytoplasmic organelles) contain powerful enzymes such as elastase and collagenase, which can digest bacterial products. However,

TABLE 10.2 Bacteria most commonly associated with periodontal disease

Porphyromonas gingivalis
Prevotella intermedia
Actinobacillus actinomycetemcomitans
Fusobacteria
Treponema spp.

these substances may also be released into the gingival tissues, causing localized tissue damage.

Much interest has focused on the chemical mediators of inflammation and on both bacterial and host-derived enzymes as potential markers of active periodontal destruction.

Genetic factors and periodontitis

There is currently interest in the contribution of host genetics to the development of periodontitis. Studies suggest that there is a host genetic component of susceptibility for several rare forms of syndromic periodontitis associated with conditions such as Papillon–Lefèvre, Ehlers–Danlos or Chédiak–Higashi syndromes. With the exception of these syndromic forms of periodontitis, specific genes have either not yet been identified or rigorously demonstrated to have a causal relationship with commonly occurring forms of periodontitis. There is no evidence of any simple pattern of genetic transmission that would support an aetiological role for a single gene mutation in chronic periodontitis.

Smoking

Cigarette smoking is a significant risk factor for both periodontitis and acute necrotizing ulcerative gingivitis (ANUG). Several mechanisms have been suggested as an explanation of how smoking predisposes to periodontal disease. The vasoconstrictive effects of nicotine may interfere with host response mechanisms and with both neutrophil and fibroblast function. Smoking may also influence the microbial composition of dental plaque. Those who smoke are 2.5–4 times more likely to develop periodontitis. Smokers also respond less well to periodontal therapy. There is good evidence that smokers experience significantly less reduction in pocket depths following periodontal therapy than non-smokers. Studies show that probing depth reduction and clinical attachment level improvements in smokers are 50% to 75% those of non-smokers following non-surgical and surgical periodontal therapy.

Stopping smoking benefits periodontal health and improves the outcome of treatment. Members of the dental team have a key role to play in educating patients on the effects of smoking on oral health and on the benefits of smoking cessation (p. 163).

Systemic disease and periodontitis

Systemic disease and periodontitis may interact in two ways. Systemic disease may predispose to periodontitis. This is an important and established factor in the aetiology of periodontitis. Systemic diseases that impair the host defence mechanism may increase the risk of periodontitis. Many systemic conditions associated with or predisposing to periodontal attachment loss have, as a common attribute, defective neutrophil function, e.g. agranulocytosis, cyclic neutropenia. Patients with uncontrolled diabetes, Down syndrome and human immunodeficiency virus (HIV) are all at increased risk of periodontitis.

The second aspect of the relationship between periodontitis and systemic disease focuses on periodontitis as a predisposing factor for systemic disorders. Research has suggested that periodontitis may predispose to cardiac disease and preterm low birth weight babies, though studies on these topics have given conflicting results and the significance of these relationships awaits determination.

CLASSIFICATION OF PERIODONTAL DISEASES

Numerous classifications of periodontal diseases have been suggested. That in most common usage is described in Table 10.3.[1]

Gingival diseases

These are divided into those conditions which are either dental plaque induced or not primarily associated with dental plaque.

Examples of plaque-induced gingivitis include gingivitis with or without local contributing factors such as tooth anatomical factors, dental restorations and appliances. General factors contributing to gingivitis include hormonal changes in puberty and pregnancy.

TABLE 10.3 Classification of diseases that may affect the periodontium

I Gingival diseases
  A Dental plaque-induced gingival diseases
  B Non-plaque-induced gingival diseases
II Chronic periodontitis
  A Localized
  B Generalized
III Aggressive periodontitis
  A. Localized
  B Generalized
IV Periodontitis as a manifestation of systemic disease
V Necrotizing periodontal diseases
VI Abscesses of the periodontium
VII Periodontitis associated with endodontic lesions
VIII Developmental or acquired deformities and conditions

Conditions not primarily associated with dental plaque include bacterial and viral infections directly affecting the gingival tissues, e.g. primary herpetic gingivostomatitis.

Chronic periodontitis

Replacing the term 'adult periodontitis', this term describes the most commonly presenting form of periodontitis. It may be localized affecting just some teeth or tooth sites, or more generalized. The term chronic periodontitis does not imply that the condition is resistant to treatment.

Aggressive periodontitis

These forms of periodontitis represent highly aggressive forms of the disease with extensive destruction of the tooth-supporting apparatus. These forms of disease were previously known as 'early-onset' periodontitis, and encompass conditions formerly known as localized juvenile periodontitis and rapidly progressive periodontitis. Two forms of aggressive periodontitis are described – localized and generalized.

Periodontitis as a manifestation of systemic diseases

This term describes periodontitis associated with underlying conditions such as blood disorders affecting the periodontal tissues or associated genetic disorders, e.g. Down syndrome, Ehlers–Danlos syndrome and Chédiak–Higashi syndrome.

However, neither diabetes nor smoking is included in this section of the classification system as they are considered to act as modifiers of all forms of periodontal disease.

Necroting periodontal diseases

These include necrotizing ulcerative gingivitis (p. 224) and necrotizing ulcerative periodontitis.

EXAMINATION AND DIAGNOSIS

The features of taking a history and examination described in Chapter 2 are relevant. However, the specific features relating to the diagnosis of periodontal disease should be noted and recorded (Table 10.4).

TABLE 10.4 Features to be noted during a periodontal examination

Visual	Deposits – supragingival plaque, calculus Gingivae – erythema, hyperplasia, recession Occlusal abnormalities
Probing	Pocket depths Bleeding on probing Subgingival calculus Furcation defects
Palpation	Mobility

EXAMINATION

Oral hygiene

Determination of the patient's oral hygiene status is crucial. The presence of supragingival plaque and calculus deposits should be noted in terms of distribution within the mouth and the quantity present. However, whilst plaque and calculus deposits give some indication of the patient's level of oral hygiene, the presence of gingival inflammation (gingivitis) provides a better indication of the patient's long-term plaque control.

Plaque When present in small amounts, plaque is not always apparent solely by visual examination, and it is necessary to run a probe around the gingival margin to enable trace amounts to be visualized. Use of a probe is also required to detect plaque in interproximal spaces.

Calculus Formed by mineralization of plaque, this is described according to its relation to the gingival margin.

Supragingival calculus Formed above the gingival margin, it accumulates most readily adjacent to the orifices of the major salivary glands: on the lingual aspects of the lower incisor teeth, and the buccal aspect of maxillary molars. It is light yellow in colour and is best visualized by drying the teeth. It is composed of calcium phosphate crystals – brushite, whitlockite, octacalcium phosphate and hydroxyapatite. It is relatively easily removed by scaling.

Subgingival calculus Dark brown or green in colour, subgingival calculus is located beneath the gingival margin. Present either in sheets covering the entire root surface or in discrete clumps, it is detectable only by tactile means or by using an air syringe to deflect the gingivae. The ball end of the CPITN probe (p. 209), used with light touch, is the most effective means of determining the location of subgingival calculus, which appears as a rough area or catch on the root surface. A sound knowledge of root surface anatomy is required to enable accurate probing. Subgingival calculus may come to lie supragingivally following gingival recession. It is firmly adherent to the root surface and its removal requires careful scaling technique.

Gingival health The condition of the gingivae should be recorded. This should take account of the distribution and severity of:

Erythema The degree of redness, an indication of gingivitis. Note areas of haemorrhage.
Hyperplasia Any gingival swellings, or areas of overgrowth.
Recession Measured as the position in millimetres of the gingival margin below the amelocemental junction.

Pocket depths A periodontal probe should be used to determine pocket depths. A note should be made of the teeth and surfaces affected and can be recorded as the average and worst depths. The patient's periodontal status can be summarized using the Basic Periodontal Examination (BPE), page 208.

Bleeding on probing The presence of bleeding on probing is a crucial indicator in periodontal examination and diagnosis, as it is the most reliable indicator of disease activity. Careful note should be made of the pockets that bleed on probing and the severity of the haemorrhage.

Furcation lesions Probing also enables furcation lesions to be detected. A furcation lesion occurs in multirooted teeth when the loss of periodontal attachment has reached the area where the roots diverge. When this occurs, it becomes increasingly difficult to ensure that all subgingival deposits are removed from the furcation and for the patient to keep this area clean.

Furcation lesions can be graded as:

Grade 1 Bone loss up to one-third of the tooth width, i.e. about 3 mm of horizontal attachment loss.
Grade 2 Bone loss between one-third and two-thirds of the tooth width.
Grade 3 A 'through and through' lesion.

Tooth mobility All teeth should be carefully palpated to determine mobility. Move the tooth between an instrument handle and an index finger in a buccolingual direction (and mesiodistal direction when no adjacent tooth is present). The amplitude of movement of the crown tip from its most extreme lingual (or distal) position should be observed.

Visible mobility should be scored as follows (Miller's Index):

Score 1 Mobility up to 1 mm.
Score 2 Mobility of 1–2 mm.
Score 3 Mobility over 2 mm and/or rotation or depression.

Occlusion Any abnormalities of occlusion should be noted. Features to be observed include: • premature contacts • overerupted teeth • deep traumatic overbites.

DIAGNOSIS

Having recorded these factors, together with information on the patient's presenting complaints, age, medical and social history, a provisional diagnosis can be made.

Additional information may be obtained from: • radiographic examination • pulp vitality tests • study models • biopsy • microbiological and haematological investigations.

Radiographs in the diagnosis of periodontal disease

Radiographs are essential in the diagnosis and management of periodontal disease, but, as with all radiographs, should be used only to supplement clinical examination (Chapter 4).

The following information should be noted:

Bone levels • degree of bone loss – mild, moderate or severe • distribution of bone loss – localized or generalized • pattern of bone loss – vertical, horizontal or both.

Furcation defects • teeth affected.

Crown : root ratio • relative length of the root to the crown – particularly relevant in incisors.

Root morphology

Restorations • overhanging margins • deficient margins • recurrent caries.

Periapical pathology – perio-endo lesions.

A definitive diagnosis should be made before formulating a treatment plan.

PERIODONTAL INDICES

Traditionally, oral hygiene was recorded as good, satisfactory, or poor. Currently, numerous indices are available. Many were developed for use in clinical trials and are of limited use in individual clinical treatment. The most commonly used are:

Debris index (Green & Vermillion 1964[2])
0 = No plaque
1 = Plaque covering 1/3 tooth
2 = Plaque covering 2/3 tooth
3 = Plaque totally covering tooth

Plaque index (Silness & Loe 1964[3])
0 = No plaque detected
1 = Looks clean but material can be removed from gingival 1/3 with probe
2 = Visible plaque
3 = Tooth covered with abundant plaque

Modified gingival index (Loe 1967[4])
0 = Healthy gingivae
1 = Gingivae look inflamed, but don't bleed when probed
2 = Gingivae look inflamed and bleed when probed
3 = Ulceration and spontaneous bleeding

THE BASIC PERIODONTAL EXAMINATION (BPE)

The Basic Periodontal Examination (BPE) is derived from the Community Periodontal Index of Treatment Needs (CPITN), which was developed as a screening tool to enable the prevalence of periodontal disease in a community to be summarized.

The index comprises six codes (0–4 and *). An individual patient's periodontal status can be summarized by six numbers. For examination purposes the dentition is divided into sextants as shown below:

17–14	13–23	24–27
47–44	43–33	34–37

The use of a CPITN probe (Figure 10.2) is mandatory. This has a ball end 0.5 mm in diameter. A colour-coded area extends from 3.5–5.5 mm. A probing force in the order of 20–25 g is recommended. This corresponds to gentle pressure; pain during probing indicates that too much force is being applied.

The CPITN probe is gently inserted into the gingival pocket and the depth of penetration read against the colour-coded band. At least six points on each tooth should be examined by gently 'walking the probe' around the tooth.

For each sextant only the highest score is recorded. Any sextant containing only one tooth is recorded as missing and the score for that tooth included in the adjacent sextant.

A simple box chart is used to record the score for each sextant.

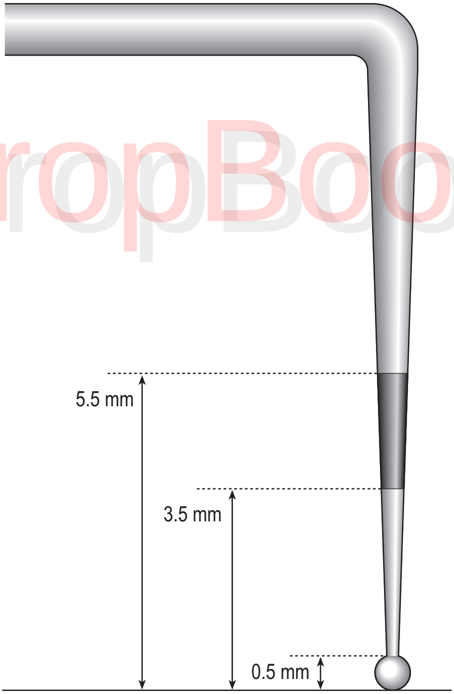


Figure 10.2 Community Periodontal Index of Treatment Needs (CPITN) probe.

BPE codes

Code 0	Healthy gingival tissues with no bleeding after probing.
Code 1	Bleeding on probing, plaque present, but no calculus or defective restoration margins, pockets <3.5 mm.
Code 2	Bleeding on probing, calculus detected or defective restoration margins but pockets <3.5 mm.
Code 3	Pocket within the colour-coded area, i.e. pocket >3.5 mm–<5.5 mm.
Code 4	Colour-coded area disappears, indicating pocket >5.5 mm.
Code *	Denotes the presence of furcation involvement or attachment loss >7 mm.

The BPE, whilst useful as a guide to a patient's overall periodontal status, is insufficiently sensitive for monitoring individual patient treatment over a period of time because only the worst score is recorded in each sextant. Codes for Periodontal Treatment Assessment have been proposed. These categorize a patient's periodontal treatment needs into three levels of complexity and are designed as a guide to the need for specialist periodontal care. In summary BPE codes 1–3 are deemed suitable for non-specialist periodontal care. Code 4 and * frequently suggest that the complexity is such that the patient may need referral for specialist periodontal care. This is particularly so if the patient is young and suffering from aggressive forms of periodontitis, or has a complicating medical history, e.g. uncontrolled diabetes.

POCKET CHARTS

The data resulting from a comprehensive periodontal examination can be recorded on a pocket chart. This usually includes the following details: • missing teeth • position of the gingival margin • pocket depth • loss of attachment • bleeding on probing • furcation lesions • mobile teeth.

The information recorded at the initial visit is important in diagnosis, treatment planning and determining prognosis. The chartings recorded during and after hygiene phase therapy indicate treatment success and highlight specific problems or areas requiring further therapy.

Other periodontal terms

False pocket Where increased probing depth is due to gingival enlargement without loss of attachment of the periodontal ligament.

True pocket Where increased probing depth results from destruction of the periodontal ligament.

Recession Where the gingival margin lies apical to the amelocemental junction.

CAUSES OF GINGIVAL RECESSION

- As a natural consequence of ageing.
- Due to plaque-induced gingival inflammation.
- Prominent position of tooth in the dental arch, e.g. buccal aspect of upper canine.
- Underlying bony dehiscence.
- Trauma:
  - toothbrushing
  - deep overbite
  - denture-induced trauma
  - other habit.
- High fraenal attachment.
- Following periodontal treatment.

STAGES IN PERIODONTAL THERAPY

Following examination and diagnosis, periodontal therapy is usually carried out in the following stages.

1. **Hygiene phase**
   This stage of therapy comprises:
   - dental health education and instruction in plaque control
   - elimination of obstacles to effective oral hygiene, including defective restoration margins, grossly malpositioned teeth and teeth of hopeless prognosis
   - non-surgical subgingival scaling and root planing.
2. **Reassessment**
   Conducted 4–6 weeks after the completion of hygiene phase. A further full periodontal assessment is carried out including plaque, gingivitis and pocket charts. This indicates the success of therapy and highlights specific problems or areas requiring further therapy.

3. **Corrective phase**
   Disease that persists following the hygiene phase is treated by further root planing or surgery.
4. **Supportive periodontal care/maintenance phase**
   Long-term follow-up is required to monitor the success of therapy. Patients should be reviewed at regular intervals to monitor oral hygiene and permit early detection of recurrent disease. Careful probing of all tooth surfaces is required to detect bleeding pockets, the only currently reliable indicator of active periodontitis.

HYGIENE PHASE THERAPY

ORAL HYGIENE INSTRUCTION

Successful prevention and treatment of periodontal disease is heavily dependent on the ability of the patient to maintain an adequate standard of plaque control. Thus the ability to motivate the patient to improve oral hygiene is of paramount importance.

Oral hygiene instruction should be tailored to the circumstances and needs of the individual patient.

Begin by explaining the nature of periodontal disease, the role of dental plaque and the need to improve cleaning in areas particularly difficult to reach, e.g. lingual aspect of lower molars.

- Models and information leaflets may be of value in aiding patient understanding.
- Visualization of plaque can be enhanced by the use of disclosing agents and a hand-held mirror.
- Rather than recommend a standardized toothbrushing regimen, it is more important to establish deficiencies in current practice and offer advice tailored to the needs of the individual patient.

Disclosing agents

Tablet For patient home use.

Solution Better for use in the surgery as solution can be used to disclose specific areas.

Toothbrushing technique

Numerous toothbrushing techniques have been described, of which the modified Bass technique is probably the simplest and most effective. The head of the toothbrush should be angled so that the filaments are at 45° to the long axis of the tooth and should be placed at the gingival margin to allow the tips of the filaments just to enter the gingival crevice. A back-and-forth movement should be used to facilitate plaque removal (Figure 10.3). It should be stressed that accessing the ‘awkward’ areas is more important than continually cleaning the more easily accessible surfaces. Scrub techniques and toothbrushes with hard filaments should be avoided as these can cause gingival recession.

The decreasing cost of electric toothbrushes has increased their popularity. While previously their use was recommended mainly for special-needs patients or those with particular dexterity problems, their use can now be more widely recommended. Powered toothbrushes have the advantage of a smaller head than conventional brushes. However, irrespective of whether patients choose to use either a manual or a powered brush, spending

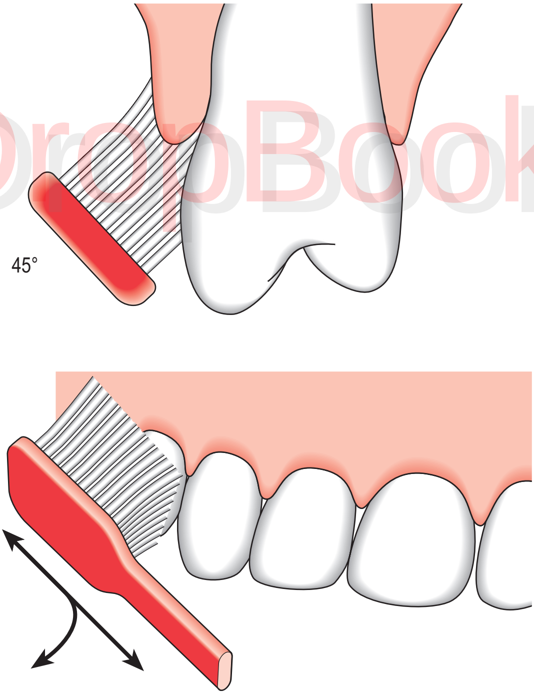


Figure 10.3 Modified Bass toothbrushing technique.

sufficient time brushing, and ensuring that the difficult-to-reach areas are clean, is the key to prevention of periodontal problems.

Interproximal plaque control Whilst plaque most readily accumulates in the interproximal region immediately beneath the contact area, this is the region least likely to be reached by toothbrush filaments. For this reason patients must also be instructed in interproximal plaque control techniques. A variety of aids for between-tooth cleaning are available (Table 10.5). Patients require individual tuition in the use of interproximal aids if they are to use them effectively.

It is however, very important not to 'overload' the patient with a vast armamentarium of tooth-cleaning devices. Only when the patient achieves a satisfactory level of toothbrushing should interproximal aids be introduced. When promoting improved plaque control, it is important to warn patients that, initially, bleeding on cleaning will be apparent due to existing gingivitis, but that this will decrease rapidly as plaque control improves. This is important as, without this advice, the treatment may appear only to worsen the presenting complaint!

TABLE 10.5 Oral hygiene aids

Toothbrushes	The most important features are the size of the head, medium texture filaments and rounded ends to the bristles
Electric toothbrushes	May be of particular benefit in patients with reduced manual dexterity
Single tufted brushes	Cleaning around lone-standing teeth, partially erupted third molars, proximal spaces adjacent to saddle areas in partially dentate patients. Also useful in localized areas of recession and exposed portions of dental implants
Floss	Waxed or unwaxed. Waxed may be easier for first-time users
Tape	Broader than floss, it passes between teeth more easily. May be beneficial where interproximal restorations are present
Superfloss	Used for cleaning under bridge pontics
Floss threader	Used to pass floss beneath pontics, cheaper than using Superfloss
Interproximal (bottle) brushes	The method of choice for interdental cleaning when space permits. Available in a range of sizes; choose the largest size which passes between the teeth without causing discomfort
Wooden sticks	Not as effective as interproximal brushes

CHEMICAL PLAQUE CONTROL

Whilst periodontal disease is bacterial in origin, the use of chemicals and drugs in the prevention of plaque accumulation is of limited use in the long-term inhibition of periodontal disease.

Reasons for chemicals being of limited use include:

- non-specific plaque hypothesis – unlike other diseases of bacterial origin, periodontal disease is caused by a range of bacterial species
- the organisms involved are commensal in origin and thus there is no logical end point to treatment
- the organisms are located subgingivally where mouthwashes do not penetrate
- many agents lack sufficient substantivity and do not remain active long enough to exert a pharmacological effect.

Other concerns relate to the indiscriminate use of antimicrobials and the contribution this may make to increasing microbial resistance to therapeutic agents.

Long-term prevention of periodontal disease is dependent on adequate mechanical plaque control.

A range of mouthwashes are available for the chemical control of plaque. However, their use should be regarded as short term until the patient is able to reintroduce or master adequate mechanical plaque control. The most effective antiplaque agent is *chlorhexidine gluconate*.

Chlorhexidine gluconate

Indications for use

- After oral surgery to reduce postoperative infection, pain and inflammation.
- In the management of periodontal problems as part of a palliative care programme.
- To help prevent drug-induced gingival overgrowth.
- For patients with fixed intraoral appliances, e.g. intermaxillary fixation devices.
- To prevent secondary infection in ulcerative conditions, e.g. ANUG, viral infection.

Other chemicals commonly used as antiplaque agents include quaternary ammonium compounds, e.g. cetylpyridinium chloride

(CPC) • phenols • triclosan – a *bis*-phenol, non-ionic germicide. Used in both mouthwashes and toothpastes, its activity can be enhanced by combination with zinc citrate or incorporation of copolymers.

SCALING AND ROOT PLANING

Root planing involves removal of subgingival plaque, calculus and infected cementum.

Removal of both sub- and supragingival deposits are essential elements of hygiene phase therapy, a process known as scaling and root planing. This can be achieved using either hand instruments or ultrasonic scalers. It is important that instruments are used correctly to ensure a smooth root surface.

Hand instruments

Scaling instruments can be classified as chisels, sickles, hoes, curettes and files (Figure 10.4). However, within these fundamental groups a wide variety of instruments are available. Scalers are usually paired, whereby one instrument is the mirror image of its partner, to ensure that the cutting edge of the scaler conforms to the tooth curvature at all times (Table 10.6).

The blade of the scaler is made of either stainless steel or tungsten carbide. To be effective, instruments should be sharpened regularly. Stainless steel instruments are sharpened using an Arkansas stone; for tungsten carbide instruments a diamond file is used. Blunt scalers may fail to remove subgingival calculus and only burnish it smooth on the root surface, making subsequent detection and removal difficult.

A modified pen grasp should be used – the scaler is grasped between the thumb and forefinger and the shank of the instrument is rested against the middle finger, which acts as the finger rest and also as the fulcrum around which the force is applied. This should be placed on tooth or bone close to the point of application of the force.

When scaling: • great care must be taken not to lacerate the soft tissues • avoid gouging or creating grooves on the root surface, which allow proliferation of subgingival plaque and decrease the chances of healing • a systematic approach to scaling is required to ensure that all tooth surfaces are cleaned.

The recommended operator and patient positions when using hand instruments are described in Table 10.7.

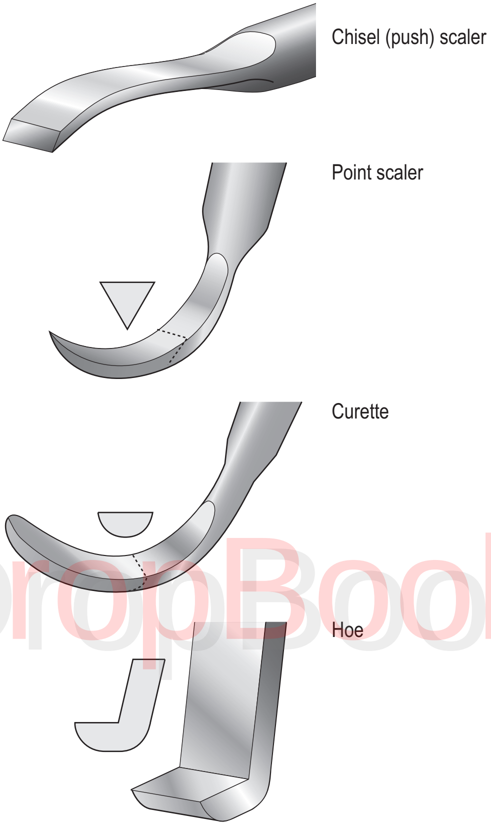


Figure 10.4 Scaling instruments.

Ultrasonic scalers

Ultrasonic scalers are composed of an electric power generator, a handpiece and an insert with a working tip. Electrical energy is converted to mechanical energy, causing the tip of the instrument to oscillate about 25 000 times per second through an amplitude of 0.006–0.1 mm. A fine water jet is directed onto the vibrating tip, not only acting as a coolant but also causing the formation and violent collapse of microscopic bubbles (the cavitation effect). The scaling action results from both the mechanical movement of the tip and the flushing and cavitation effect of the water spray.

TABLE 10.6 Scaling instruments

Instrument	*Description*	*Area of application*
Chisel (push) scaler	Chisel head, angled at 45°	Applied from labial aspect to remove gross supragingival deposits from approximal surfaces of mandibular anterior teeth
Point scaler (includes sickle scalers and jacquettes) (also known as trihedral scalers)	Triangular in cross-section and have two cutting edges which converge to a sharp point	All buccal and lingual embrasure areas, supragingival and 2–3 mm subgingival Manipulated with wrist movement so that the point always moves towards and into the embrasure
Hoes	Have a blade set at 100° angle to the shank and cutting edge is beveled at 45° A set of 4 is required to give access to all tooth surfaces	Allows access to deep pockets. Used in a series of short pull strokes to remove subgingival deposits
Curettes	Curved spoon-shaped blade with two cutting edges that meet to form a rounded toe	Supra- and, especially, subgingivally throughout the mouth. Used following hoes in root planing
Periodontal files (Hirschfeld files)	Has multiple straight cutting edges at the working end of the instrument	Used to roughen the surface of burnished calculus, or to ‘crush’ calculus to facilitate its removal with other instruments

A variety of working tips are available. Ultrasonic scalers are best used for removing gross supragingival deposits and heavy staining and can also be used subgingivally.

As much of the instrument tip as possible should be held in contact with the tooth (to avoid gouging), and should be kept in motion at all times with a light brushing stroke. The lowest possible power setting consistent with satisfactory resonance should be used. Efficient high-volume aspiration is essential when using an ultrasonic scaler to remove the water and reduce aerosol production.

TABLE 10.7 Scaling technique – operator and patient positions

Teeth	*Operator position (12 o'clock = directly behind patient)*	*Patient position*
	7 o'clock, facing patient	Semi-upright, patient's head between operator's shoulder and elbow
33–43 buccal	Direct vision	
33–43 lingual	Reflected vision	
	11 o'clock, behind patient	Patient supine
34–38 lingual	Direct vision	
44–48 buccal	Direct vision	
34–38 buccal	Direct vision	
44–48 lingual	Direct vision	
13–23 buccal	Direct vision	
13–23 palatal	Reflected vision	
14–18 buccal	Direct vision	
14–18 palatal	Reflected vision	
24–28 buccal	Direct vision	
24–28 palatal	Direct and reflected vision	

Avoid ultrasonic scalers in patients fitted with a cardiac pacemaker.

Healing following scaling and root planing

Reduction in pocket depth occurs by two principal mechanisms. • Recession of the gingival margin due to resolution of inflammation and subsequent reduction in swelling and hyperplasia. • Reattachment to the root surface. This occurs primarily by the formation of a long junctional epithelial attachment. Epithelial cells grow from the gingival sulcus to repopulate the pocket lining and attach by hemidesmosomes to the root surface. This is most likely to occur in the absence of inflammation; hence the need for scrupulous plaque control and complete removal of deposits from the tooth root to leave a smooth, plaque- and calculus-free surface.

Whilst the periodontal ligament contains precursor cells that have the ability to form a connective tissue reattachment, the restitution of a true periodontal ligament following treatment occurs only in limited circumstances. This may be encouraged by the use of guided-tissue regenerative techniques (p. 230).

Polishing

Well-polished, smooth tooth surfaces retain plaque less well than rough surfaces and thus it is useful to polish teeth after removal of plaque and calculus. • Gross stains should be removed using hand or ultrasonic instrumentation. • A small webbed rubber cup held in a special contra-angled handpiece and run at slow speed should be used. • The polishing cup is placed above the gingival margin and smoothly pulled coronally. • Do not repeatedly polish the same tooth surface as it is important to avoid overheating. • Use a fluoride-containing paste.

An often overlooked benefit of polishing teeth is the sense of well-being it promotes in the patient, thereby providing a greater incentive to carry out home care procedures.

PERIODONTAL SURGERY

Periodontal surgery can be defined as any procedure involving the periodontal tissues requiring the use of a scalpel.

A wide variety of surgical techniques and procedures have been described. Periodontal surgery aims to reduce pocket depth and/or restore gingival contour to a state that can be maintained free of inflammation by the patient.

When is periodontal surgery carried out? Periodontal surgery should ideally be performed only after hygiene phase therapy has been completed. There are several reasons for this. • When pockets are due mainly to gingival hyperplasia, improvement in oral hygiene may result in resolution of inflammation and reduction in swelling, so surgery is avoided. • Reduction in inflammation means that haemorrhage at operation is less of a problem. • Where pockets are shallow (<5 mm), resolution following scaling and root planing may obviate the need for surgery. • Flap surgery is contraindicated in patients who are unable to maintain adequate levels of plaque control or who continue to smoke tobacco.

Periodontal surgery can be classified as gingivectomy, flap procedures or mucogingival surgery.

GINGIVECTOMY

Gingivectomy involves the excision of excess gingival tissue.

Indications Gingival hyperplasia • epulides, pericoronal flap • elimination of subgingival restoration margins • recontouring following acute necrotizing ulcerative gingivitis.

Technique

- The depth and morphology of the pockets or tissues to be eliminated are established by probing and can be marked using pocket-marking tweezers.
- Excess tissue to be removed is excised in toto by an incision made at an angle of 45° to the long axis of the tooth, so that the blade impacts against the tooth slightly apical to the base of the pocket.
- Sterile swabs are used to control haemorrhage before a periodontal dressing (e.g. CoePack®) is placed to protect the wound area.

FLAP PROCEDURES

Involve the elevation of a flap to permit access to the root surface.

The basic principles of flap design must be followed: • must be sufficiently large to expose any underlying bone defects • base must be wide enough to maintain adequate blood supply • incisions must allow movement of the flap without tension • important vessels and nerves must be preserved.

There are two basic types: replaced flap and repositioned flap.

Replaced flap

Here the flap is sutured in or near its original position. Also known as open flap curettage or modified Widman flap.

Indications • Allow access to root and alveolar bone to enable removal of subgingival deposits and granulation tissue where pockets persist following hygiene phase therapy. • Reduce pockets by encouraging a new epithelial and/or connective tissue reattachment at a more coronal level. • Used in osseous regenerative procedures such as guided tissue regeneration.

Technique

- An intracrevicular incision is made around the necks of the teeth with the scalpel parallel to the long axis of the teeth. Care should be taken to preserve as much tissue as possible. (In the modified Widman flap, the pocket-lining epithelium is also excised via an inverse bevel incision.)
- Vertical relieving incisions are required buccally if pockets are deep. Flaps should be sufficiently extensive to expose the marginal bone.
- Subgingival calculus deposits and granulation tissue should be removed.
- The flaps are replaced with interrupted sutures, taking care to achieve approximation of wound edges interdentally where possible.

Repositioned flap

Here, the flap is repositioned apically, coronally or laterally following surgery. Apical repositioning is the most common.

Indications • Eliminate pockets by positioning the gingival tissue apically • Increase the zone of keratinized gingivae. • Expose additional root for restorative procedures, i.e. crown lengthening.

Technique

- Buccal and lingual/palatal flaps are raised using either intracrevicular or inverse bevel incisions.
- The flap is reflected and dissected from the alveolar process in order that it is sufficiently mobile to enable repositioning.
- Any adherent granulation tissue or subgingival calculus deposits are removed.
- The flap is sutured in the desired position and may be held in place by the placement of a periodontal pack.

MUCOGINGIVAL SURGERY

Procedures that involve alteration of mucogingival relationships.

Are concerned with: • increasing the width of attached gingivae • eliminating pull on the free gingival margin by fraenulae or muscle pull • covering gingival clefts or recession.

Unlike the alveolar mucosa, the attached gingivae are covered by stratified squamous epithelium and thus are designed to withstand frictional forces encountered in eating, toothbrushing, etc. Whilst in the past it was thought that a minimal width of attached gingivae was necessary to prevent periodontal disease, it is now accepted that the presence of the attached gingivae is not crucial for the maintenance of gingival health, provided the patient can keep the area clean. Thus mucogingival procedures are carried out less frequently than in the past.

Techniques can be classified as:

Fraenectomy

Involves excision of the fraenum or high muscle attachments and is used: • to dissipate pull on the gingival tissues and prevent recession • to enable easier access for tooth cleaning • before orthodontic treatment to enable approximation of upper central incisors where an interdental fraenal attachment may prevent closure of a midline diastema.

Laterally repositioned flaps

Used to cover isolated areas of recession.

Free gingival grafts

Used to cover isolated areas of recession.

Care and instructions following periodontal surgery

Crucial to the success of all periodontal surgical procedures is the maintenance of a plaque-free environment during the healing process.

In order that sutures or periodontal dressings are not disturbed in the immediate postoperative period, mechanical plaque control in the area of surgery should be suspended and replaced with the twice-daily use of 0.2% chlorhexidine gluconate mouthwash. Patients should be encouraged to rinse vigorously, ensuring the solution reaches the interdental areas. Patients should be advised to use appropriate analgesics (paracetamol or ibuprofen) to relieve postoperative discomfort. Alcohol, very hot drinks, or excessive exertion should be avoided in the 24 hours following surgery.

Sutures and any packs or dressings are normally removed 1 week postoperatively. The use of chlorhexidine should be encouraged for a further week to 10 days, before mechanical interproximal cleaning is reintroduced.

Patients should be reviewed at regular intervals to monitor healing and reinforce the need for scrupulous plaque control.

Complications of periodontal surgery

Periodontal surgery almost invariably results in a degree of gingival recession. This has a number of consequences: • the increased exposure of the root surface may lead to dentine hypersensitivity • the increased tooth length may have implications for aesthetics in anterior teeth • the exposed root surface is more vulnerable to root caries.

RECALL MAINTENANCE

When active periodontal therapy is complete, a recall and maintenance programme is required. This aims to monitor plaque control and check for recurrent periodontal disease, best indicated by bleeding on probing. In general, recall intervals should initially be short (6–8 weeks) and then gradually extended provided the periodontium remains stable. The precise recall interval should be tailored to the patient's individual circumstances.

ACUTE PERIODONTAL CONDITIONS

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

An acute ulcerative condition affecting the marginal gingivae.

Is painful, of sudden onset and leads to loss of the interdental papillae.

Clinical features: • Interproximal necrosis – begins at tips of the papillae; may spread laterally to involve all of gingival margin

• ulceration – grey slough, easily removed to leave raw bleeding surface • pain • halitosis • metallic taste. If severe: • lymphadenopathy • fever • malaise.

Site • Most commonly affected sites are incisor region and partially erupted third molars.

Occurrence: • Second and third decades (mostly 18–26 year olds) • males = females • more common in winter.

Predisposing factors: • Inadequate oral hygiene • smoking • stress • impaired host resistance.

Diagnosis Usually on clinical grounds but can be confirmed by a smear which shows • neutrophils • spirochaetes • fusiforms.

Treatment

Immediate

Local treatment • Mechanical debridement; ultrasonic scaler will remove debris and has the advantage of oxygenating area • oral hygiene instruction.

Systemic treatment • Metronidazole, especially if fever or pyrexia present; 200 mg three times daily for 3 days.

Follow-up treatment

Further investigations are indicated if ANUG fails to resolve or patient suffers recurrent bouts of ANUG. May indicate underlying immunosuppression (e.g. HIV). Occasionally gingival recontouring is required to correct gingival morphology and eliminate interdental craters.

PERIODONTAL ABSCESS

An acute collection of pus within a gingival or periodontal pocket.

Predisposing factors The presence of subgingival plaque; also frequently associated with a deep periodontal pocket. Clinically requires differentiation from an abscess, which is pulpal in origin (a periapical abscess). Deduced from clinical features, radiographs and vitality tests (Table 10.8). Diagnosis is complicated by heavily restored or multirooted teeth.

Remember, can originate from both periapical and periodontal sources – a combined *perio-endo lesion*.

TABLE 10.8 The differentiation of abscesses that are periodontal and periapical in origin

	Periodontal abscess	*Periapical (dental) abscess*
Pain	Acute onset	History of toothache
Swelling	Usually localized, extraoral swelling unlikely	Over tooth apex, more likely to be extensive
Pocket	Always present; more likely in presence of periodontal disease	± pocket
Sinus	Frequently on attached gingivae	Tracks to apex
Percussion	TTP, worse laterally	TTP, especially in axial direction
Restoration status	More likely if tooth caries-free/unrestored	More likely in heavily restored tooth
Vitality	Tooth vital	Tooth non-vital
Radiographs	Little evidence in early stages; bone loss	Loss of lamina dura in periapical area (after 10 days or so)

TTP, tender to percussion.

Treatment of periodontal abscess

1. Establish drainage
   a. most frequently via gingival sulcus
   b. occasionally requires incision if fluctuant
2. Microbiological sample for culture and sensitivity
3. Subgingival scaling to remove
   a. foreign objects
   b. subgingival calculus (there is evidence that restricting scaling to the superficial aspects of the pocket results in better healing – periodontal ligament fibres are not destroyed)
4. Irrigate with sterile saline or chlorhexidine
5. If drainage can be achieved via the the periodontal pocket, the systemic antibiotics/antimicrobials are often unnecessary. However, if systemic involvement, e.g. cellulitis, pyrexia: amoxicillin 250 mg three times

daily for 7 days or metronidazole 200 mg three times daily for 5 days should be prescribed.
6. Review in 2–3 days
7. Treatment for underlying problem.

PERIO-ENDO LESION

Occurs when an infection involves both pulp and periodontal ligament space simultaneously.

May arise as a result of: • infection in a necrotic pulp draining via the periodontal ligament (usually in the presence of existing periodontal disease) • toxins from pulp reaching periodontal ligament via lateral or accessory canals, especially in the furcation region • the root having been perforated by a pin or post.

Treatment Endodontic treatment should be carried out *before* periodontal treatment, as resolution of the endodontic problem is a necessary prerequisite for periodontal healing.

ANTIBIOTICS IN THE MANAGEMENT OF PERIODONTAL DISEASE

Whilst antibiotics are beneficial in the management of acute periodontal infections, they have a limited role in the long-term treatment of periodontitis. The non-specific nature of the bacteria that cause chronic periodontitis, the rapid repopulation of periodontal pockets following therapy and the fact that most periodontopathogens can be found in healthy mouths mean that systemic antibiotics are not indicated in the routine management of chronic periodontitis.

When used, antibiotics can be given *systemically* or delivered *locally*.

Systemic antimicrobials Prescription of a tetracycline in cases of aggressive early-onset forms of periodontitis has been advocated. Some authorities have advocated a combined regimen of metronidazole and amoxicillin.

Locally delivered antimicrobials designed for direct insertion into the periodontal pocket are available. They consist of acrylic fibres or gel impregnated with either a tetracycline or metronidazole. A chlorhexidine-impregated chip (PerioChip®) is also available for local insertion in a periodontal pocket. These devices may be of some benefit in persistent, isolated pockets, in patients with good oral hygiene. They should be used in conjunction with root planing.

Concerns over increasing microbial resistance to antibiotics means very careful consideration should be given to the potential benefits in a given case before resorting to antimicrobial therapy.

Host modulatory therapy In addition to their bacteriostatic effects, tetracyclines are known to inhibit proteolytic enzymes such as collagenase. This has led to their use in subantimicrobial doses as an adjunct to scaling and root planing (Periostat®). The recommended dose of, e.g., doxycycline is 20 mg twice daily for 3 months up to a maximum of 9 months of continuous dosing. At this dose the doxycycline is said to limit the effects of collagenase in periodontal breakdown, but must be used in conjunction with scaling and root surface debridement of the highest standard.

OCCLUSION AND PERIODONTAL DISEASES

A primary function of the periodontal tissues is to support the tooth when a load is applied. If subjected to excessive force, resorption of the surrounding alveolar bone may ensue.

Contrary to previous suggestions, trauma from occlusion cannot *induce* periodontal tissue breakdown. However, in the presence of pre-existing periodontal disease, excessive occlusal loading may increase the rate of periodontal destruction.

Occlusal trauma is damage to the supporting tissues caused by excessive occlusal stresses. This can arise from a number of sources:

Habits Bruxism, tooth grinding, pencil/fingernail chewing.
Dental treatment Lack of posterior support due to loss of molar/premolar teeth, poorly designed dentures, poorly contoured restorations, orthodontic treatment.
Occlusal disharmony Due to tooth drifting, overeruption creating interferences and preventing smooth closure to intercuspal position.
Lack of periodontal support Due to periodontal disease.
Treatment of occlusal trauma Involves removal of the excess load and concurrent treatment of the coexisting periodontal disease. This may involve selective tooth grinding, provision of a biteguard, or splint.

SPLINTS

Splint – a means of increasing tooth support by joining to an adjacent tooth or teeth.

Current indications are limited to: • tooth mobility interfering with mastication or causing patient discomfort • retaining teeth that have been realigned by orthodontic therapy.

Splints may be removable or fixed (Table 10.9).

FURCATION LESIONS

A furcation lesion describes loss of periodontal attachment extending to the bi- or trifurcation of multi-rooted teeth.

When bone loss involves the inter-radicular area, removal of subgingival deposits and subsequent maintenance by the patient can be difficult.

In addition to scaling and root planing, the following procedures may be considered necessary depending on the severity of the furcation defect.

Furcation-plasty

A flap is raised and the tooth and/or bone is recontoured to facilitate self-cleansing. Use is restricted to buccal and lingual defects. Dentine hypersensitivity may be a problem.

TABLE 10.9 Classification of splints

Removable

Partial denture – connectors give additional support to mobile teeth

Acrylic occlusal splint (or biteguard) – worn at night only

Fixed

Multistrand wire splint – attached with unfilled composite resin, this is the splint of choice. Its use may not be possible where there is a deep overbite or diastemas between the teeth

Composite strands – attached with resin

Etch-retained metal splints – e.g. Rochette and Maryland splints. Prone to failure due to inflexible nature

Root resection

Involves sectioning of the tooth and removal of one or two roots. The root to be retained should be endodontically treated and then restored with a crown or as an abutment for a double crown.

Tunnel preparation

Used in lower molars. The interdental space is widened surgically so that, when healing is complete, an interproximal brush can be passed between the roots, underneath the crown. Where space is limited, complete separation of the roots (*hemisection*) followed by placement of a double crown (where the roots are crowned and joined by an occlusal pontic) may facilitate creation of sufficient space to accommodate an interproximal hygiene aid.

Guided-tissue regeneration

This surgical technique involves elevation of a mucoperiosteal flap and curettage of granulation tissue and subgingival deposits. A polytetrafluoroethylene (PTFE) membrane (e.g. Gore-tex®) is then sutured under the flap to cover the bony defect. The membrane acts as a barrier to prevent epithelial downgrowth and allows the formation of a connective tissue reattachment and encourages bony infill of the defect. The membrane is then surgically removed 6 weeks later. Resorbable membranes (collagen, polylactid) negate the need for a re-entry procedure.

It should be remembered that successful management of furcation defects is dependent on optimal home care, and the above procedures are time consuming and technically demanding. For this reason, cases should be selected with care. Where the tooth in question is non-essential, scaling and root planing as a palliative measure may be preferable to complex treatment options.

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